due to increased plasma MSH levels consequent upon dopamine depletion (Shuster *et al.*, 1973). Drugs such as chlorpromazine which deplete or compete with brain dopamine will also induce seborrhoea (Goolamali *et al.*, 1974; Thody and Shuster, 1973; Shuster and Thody, 1974) and an increased release of MSH (Thody and Shuster, 1973), and we expect to find a similar increase in plasma MSH in patients with phenylketonuria. Recent evidence suggests that β-MSH does not occur naturally in man (Scott and Lowry, 1974) and it is likely that the immunoreactive "β-MSH" we have been measuring (Thody and Plummer, 1973) is related to β-lipotrophin. This hormone contains the MSH peptide sequence and is strongly sebotrophic (Thody and Shuster, 1971).

Our observation that in patients with phenylketonuria the S.E.R. is increased in women more than in men is difficult to explain but we have similarly noted that the severity of Parkinsonism and degree of seborrhoea is related in women but not in men (Burton *et al.*, 1973 b) even though the plasma immunoreactive "β-MSH" is increased in both sexes (Shuster *et al.*, 1973). Not too much importance should be attached to our failure to find a consistent decrease in S.E.R. after levodopa because only a few patients were given the drug and not all of these had seborrhoea and it has been shown that the decrease in S.E.R. after levodopa in Parkinsonism is related to the initial seborrhoea. The effect of levodopa requires further study both to establish the effect on the S.E.R. and because of its possible use in the management of the disease.

We thank the psychiatrists at the Prudhoe and Monkton Hospital, Northumberland, the Northgate and District Hospital, Morpeth, and the Royal Albert Hospital, Lancaster, for permission to study their patients. Thanks are also due to the Medical Research Council, the Wellcome Trust, and the North of England Cancer Campaign for grants.

References

Burton, J. L., Cartlidge, M., and Shuster, S. (1973 a). *British Journal of Dermatology*, **88**, 475.
Burton, J. L., *et al.* (1973 b). *British Journal of Dermatology*, **88**, 263.
Crome, L. (1962), *Journal of Neurology, Neurosurgery, and Psychiatry*, **25**, 149.
Cunliffe, W. J., and Shuster, S. (1969). *British Journal of Dermatology*, **81**, 697.
Fleischer, T. L., and Zeligman, I. (1960). *Archives of Dermatology*, **81**, 898.
Goolamali, S. K., *et al.* (1974). In preparation.
Hassel, C. W., and Brunsting, L. A. (1959). *Archives of Dermatology*, **79**, 458.
Knox, W. E., and Hsia, D. Y. Y. (1957). *American Journal of Medicine*, **22**, 687.
Paine, R. S. (1957). *Pediatrics*, **20**, 290.
Robbins, J. (1968). *Clinical Research*, **16**, 554.
Scott, A. P., and Lowry, P. J. (1974). *Journey of Biochemistry*, **139**, 593.
Shuster, S., and Thody, A. J. (1974). *Journal of Investigative Dermatology*, **62**, 172.
Shuster, S., *et al.* (1973). *Lancet*, **1**, 463.
Thody, A. J., and Plummer, N. A. (1973). *Journal of Endocrinology*, **58**, 263.
Thody, A. J., and Shuster, S. (1971). *Journal of Endocrinology*, **50**, 533.
Thody, A. J., and Shuster, S. (1973). *Journal of Endocrinology*, **58**, XXV.

MEDICAL MEMORANDA

Ventricular Septal Defect in a Battered Child

ALAN REES, JOHN SYMONS, MICHAEL JOSEPH, CHRISTOPHER LINCOLN

British Medical Journal, 1975, **1**, 20

Manifestations of deliberate injury to children, the so-called battered babies, have included descriptions of damage to almost every organ since the original observations of Caffey (1946). A history of repeated trauma in the home caused by one of the immediate family seems to us more important in making this diagnosis than the age of the victim. The case presented here shows clear evidence of a traumatic ventricular septal defect (V.S.D.). Reviews of the literature on this condition (Pollock *et al.*, 1952; Rubinstein and Levison, 1961; Rosenthal *et al.*, 1970; Moraes *et al.*, 1973) show the numbers to be small, children to be rarely involved, and deliberate trauma to be uncommon.

Case History

A 5-year-old girl was well until 14 days before admission to hospital. She had been under hospital observation for the first eight months of life because of low birth weight and under close review by her family doctor since then because of poor social circumstances. No heart murmur had ever been heard; she had been fit and active. Two weeks after an assault by her stepfather during which she was kicked in the chest, however, the family doctor found her to be in cardiac failure with a loud systolic murmur. She was referred to the local general hospital and then transferred to the paediatric cardiac unit at this hospital for investigation.

On admission she looked ill and had multiple bruises, though there were none on her chest or abdomen. There was a sinus tachycardia (160/min). Blood pressure was 75/40 mm Hg and the jugular venous pressure was raised. The liver was palpable 4 cm below the costal margin and there was slight oedema of the feet and ankles. A systolic thrill was felt over the anterior chest and a grade 5/6 pansystolic murmur was heard maximally at the left sternal edge at the fourth left intercostal space. The first and second sounds were normal and the third sound was present. There was tachypnoea (60/min) and fine crepitations at both lung bases.

Investigations.—Haemoglobin 10·3 g/100 ml; packed cell volume 34%; mean corpuscular haemoglobin concentration 30%; white cell count 8,500/mm^3 (normal differential); urea and electrolytes normal. *X*-ray examination showed an enlarged heart with perihilar shadowing suggestive of pulmonary oedema. The left atrium was not enlarged. E.C.G. showed sinus rhythm and a mean frontal QRS axis of +45°. There was a 3-mm P wave in lead II, indicating right atrial hypertrophy (Liebman, 1968). Otherwise the tracing was within normal limits. Echocardiography showed a left ventricular output estimated at 12 l./min/m^2. At cardiac catheterization a large V.S.D. was shown by oximetry with a pulmonary to systemic flow ratio of 8·1. Peak ventricular systolic pressures were left 75 mm Hg, right 32 mm Hg. Left ventricular cineangiography showed no mitral incompetence but a defect low in the ventricular septum.

Management.—Operation was deferred for eight weeks from the time of the injury to allow the margins to fibrose. The cardiac failure was controlled with digoxin and diuretics and the radiological signs of pulmonary oedema cleared, though pulmonary plethora remained with cardiac enlargement. The child was breathless on moderate exertion but otherwise appeared well and active. The defect was closed using cardiopulmonary bypass. There was an aneurysmal area about 2 cm by 1·5 cm in the apex of the right ventricle (fig. 1). On incising this the defect was seen in the apex of the muscular septum (fig. 2). It was relatively large, 1·5 cm in diameter, with margins composed of firm fibrous tissue. It communicated with a false chamber in the apex of the right ventricle formed partly by the aneurysm and, in a cephalad direction, by

Paediatric Department, Brompton Hospital, London SW3 6HP
ALAN REES, F.R.C.S., Senior Surgical Registrar
JOHN SYMONS, M.R.C.P., Senior Medical Registrar
MICHAEL JOSEPH, F.R.C.P., Consultant Paediatrician
CHRISTOPHER LINCOLN, F.R.C.S., Consultant Cardiac Surgeon

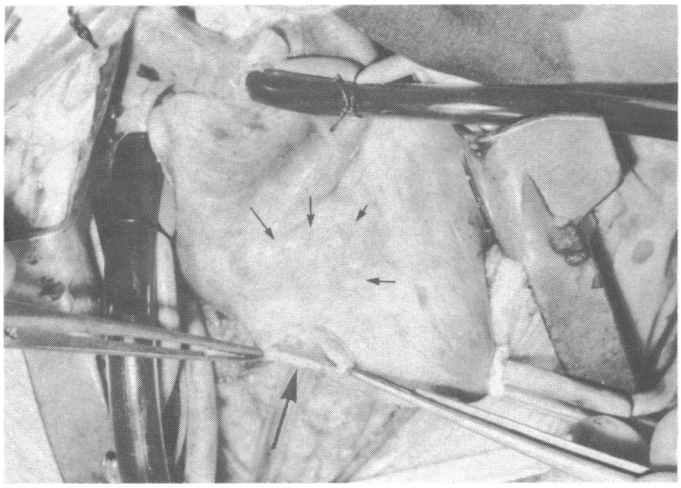

FIG. 1—Photograph taken during operation showing pale area of aneurysm in apex of right ventricle (fine arrows) and incision into aneurysm (broad arrow).

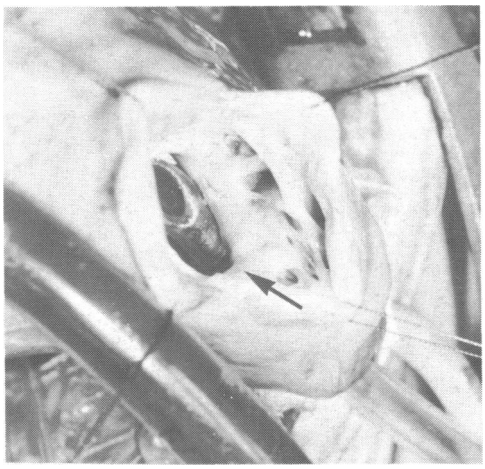

FIG. 2—Aneurysm opened widely and held open with stay sutures. Edge of V.S.D. is arrowed, with end of left ventricular vent in situ. Fenestrations are seen to right of aneurysm.

an abnormal trabeculated formation which allowed communication with the body of the right ventricle by a series of fenestrations. The defect was closed with a Dacron patch and the aneurysm was plicated with interrupted sutures. The postoperative course was uneventful and the pulmonary plethora diminished rapidly, as did the heart size.

Comment

Moraes *et al.* (1973) in a review of the literature discovered only 21 cases of surgically treated traumatic V.S.D.s. They noted that the mortality was low when surgical closure was delayed—that is, more than two months after trauma—but when it was performed as an emergency soon after the accident the mortality was much higher. As this type of injury occurs after violent trauma such as high-speed motor accidents it is likely that such cases will be seen with increasing frequency (Goggin *et al.*, 1970). In the present case, however, the trauma was a kick in the chest, which though violent cannot compare in severity with a motor accident. Presumably the pliability of the thoracic cage allowed sufficient distortion for the apex of the heart to be crushed against the vertebrae. It was notable that there was no rib fracture or any bruising of the anterior chest.

This child had suffered repeated violence from her stepfather and can therefore be placed in the battered-baby category. Skeletal injuries, lacerations, rupture of abdominal organs, blindness, and psychological damage have been recorded (Kempe *et al.*, 1962; Harcourt and Hopkins, 1971; Mushin, 1971) but no case of cardiac injury has previously been found in this condition. The knowledge that the child's heart was normal before the assault and the appearance of cardiac failure and a murmur within two weeks of the injury enabled the diagnosis of traumatic V.S.D. to be made with confidence. The position of the defect in the commonest position for traumatic V.S.D.s (Parmley *et al.*, 1958) and the presence of the overlying aneurysm was confirmatory evidence. Though reports on a number of traumatic V.S.D.s have indicated that a substantial proportion may close spontaneously (Goldfach and Wang, 1972) the haemodynamic effects of the lesion in our patient made surgical closure obligatory. The temporary control of cardia failure with digoxin and diuretics allowed time for healing of the defect margins. The child was never asymptomatic, however, and remained in hospital over this period. It has been suggested that six to eight weeks is a reasonable time for fibrosis of the margins to occur (Cleland *et al.*, 1961). The firm fibrous tissue margins of the defect found in this patient suggest this time interval to be adequate.

References

Caffey, J. (1946). *American Journal of Roentgenology*, **56**, 163.
Cleland, W. P., *et al.* (1961). *British Journal of Diseases of the Chest*, **55**, 17.
Goggin, M. J., Thompson, F. D., and Jackson, J. W. (1970). *British Medical Journal*, **2**, 767.
Goldfach, R. B., and Wang, Y. (1972). *Minnesota Medicine*, **55**, 325.
Harcourt, B., and Hopkins, D. (1971). *British Medical Journal*, **3**, 398.
Kempe, C. H., *et al.* (1962). *Journal of the American Medical Association*, **181**, 17.
Liebman, J. (1968). In *Heart Disease in Infants, Children and Adolescents*, ed. A. J. Moss, and F. H. Adams. Baltimore, Williams and Wilkins.
Moraes, C. R., *et al.* (1973). *Angiology*, **24**, 222.
Mushin, A. S. (1971). *British Medical Journal*, **3**, 402.
Parmley, L. F., Manion, W. C., and Mattingly, T. W. (1958). *Circulation*, **18**, 371.
Pollock, B. E., Markeiz, R. A., and Shuey, H. E. (1952). *American Heart Journal*, **43**, 273.
Rosenthal, A., Parisi, L. F., and Nadas, A. S. (1970). *New England Journal of Medicine*, **283**, 338.
Rubinstein, P., and Levison, D. C. (1961). *American Journal of Cardiology*, **7**, 277.